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Journal of Acute Medicine 6 (2016) 105–106

www.e-jacme.com

Letter to the Editor

Risk of Guillain–Barré syndrome from fresh chicken in the United Kingdom

Abstract

A recent survey by the Food Standards Authority indicated that nearly two-thirds of chickens sold by major retailers in the UK are contaminated with the bacterium *Campylobacter*. From a public health and neurological perspective campylobacteriosis is of great importance. Not only is it a frequent source of food poisoning, but one species, *Campylobacter jejuni*, is also known to trigger Guillain–Barré syndrome, the most common cause of flaccid paralysis worldwide. Here we briefly review the pathogenesis of Guillain–Barré syndrome and highlight clinical features of the disease.

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Keywords: *Campylobacter jejuni*; Guillain–Barré syndrome; Miller Fisher syndrome

A recent survey by the Food Standards Authority indicated that nearly two-thirds of chickens sold by major retailers in the UK are contaminated with the bacterium *Campylobacter*.[1] The survey tested 4011 samples of whole, UK-produced fresh chicken between February 2014 and February 2015. Skin contamination ranged between 66% and 80% among retailers. Worryingly, *Campylobacter* was also isolated from approximately 7% of exterior packaging. From a public health and neurological perspective campylobacteriosis is of great importance. Not only is it a frequent source of food poisoning, but one species, *Campylobacter jejuni*, is also known to trigger Guillain–Barré syndrome (GBS), the most common cause of flaccid paralysis worldwide.[2]

Campylobacter is a spiral-shaped Gram-negative bacterium and a leading cause of zoonotic disease worldwide. Most infections in humans are caused by *C. jejuni*, which is carried as a commensal organism in the gastrointestinal tracts of poultry.[3] *C. jejuni* is the most common cause of infectious diarrhea in developed countries and likely to account for many of the estimated 280,000 cases of food poisoning caused by *Campylobacter* species in the UK each year.[1] The symptoms and severity of *Campylobacter* enteritis vary considerably. Typically, onset of gastrointestinal symptoms and fever are 3–5 days after exposure. In some patients diarrhea is minimal and infection may be subclinical in up to half. Although *C. jejuni* can be cultured from stool samples up to 2 weeks after exposure, neurological symptoms may develop later, in which case serological testing is advised if confirmation is required. This is especially important if there is clustering of cases, as this may have important public health implications. Only 1 in 1000 infected patients goes on to develop GBS.[4] The exact reasons behind this remain unclear, and although some strains of *C. jejuni* are known to trigger GBS more than others,[5] host factors, including altered immune regulation are likely to be important. In a large epidemiological study, median onset of neurological symptoms attributable to GBS following exposure to *C. jejuni* was 10 days and the shortest interval was 3 days.[6]

Clinical history, including details related to meals eaten at communal functions and contact with others who have similar symptoms, are important for public health purposes.

The incidence of GBS in Western countries is approximately 1.11/100,000 person-years and increases with age.[7] GBS represents a spectrum of related disorders which vary considerably with respect to clinical phenotype and severity.[8] In its commonest and most severe form, patients present with ascending limb weakness associated with glove-and-stocking type sensory disturbance. Approximately 25% of these

http://dx.doi.org/10.1016/j.jacme.2016.09.002

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patients develop respiratory insufficiency and many require mechanical ventilation. Miller Fisher syndrome is significantly less common and associated with external ophthalmoplegia and cerebellar-like ataxia. Other rarer subtypes also exist and some patients may only develop ptosis.

GBS and Miller Fisher syndrome are considered post-infectious neuropathies. *C. jejuni* is an important trigger and has been isolated in up to one-third of cases. Much of what is known about the pathogenesis of GBS has come from studying *C. jejuni*. By a process of mistaken identity, known as molecular mimicry, cross-reactivity between surface epitopes of the bacterium *C. jejuni* and nerve gangliosides (GM1, GD1a, and GQ1b) can induce pathogenic autoantibodies, which may cause axonal-type neuropathy in some patients.[2,9] Different antiganglioside antibody profiles are thought to account for phenotypic differences between patients. For example, ganglioside GQ1b has been identified in cranial nerves III, IV, and VI and may explain why patients with Miller Fisher syndrome, which is associated primarily with anti-GQ1b antibodies, develop ophthalmoplegia.[10]

GBS is an important neurological emergency and up to 5% of patients die despite modern treatment. Timely diagnosis and commencement of appropriate treatment therefore remains essential. Intravenous immunoglobulin and plasma exchange are of proven benefit and improve outcome, especially if commenced early. Diagnosis of GBS and related disorders can be made based on clinical information alone in the majority of patients.[8] The presence of core clinical features, including history of antecedent infection, distal paraesthesias at or before onset, symmetric limb or cranial nerve weakness, monophasic disease course, and diminished deep tendon reflexes are highly suggestive of GBS and common to all subtypes. Although cerebrospinal fluid albuminocytological dissociation (raised protein and normal cell count) or neurophysiological evidence of neuropathy support diagnosis, they may be normal or nondiagnostic early in the disease course. Demonstration of serum antiganglioside antibodies also supports diagnosis but should not be relied upon or delay treatment.

News that *Campylobacter* appears to be rife among fresh chicken bought in the UK should also alert physicians to be more vigilant of GBS. Knowledge of the core clinical features of GBS and related disorders is likely to lead to early recognition and treatment of this potentially life-threatening disease. Prevention is better than cure and consumers should also be encouraged to cook their chickens appropriately.

Conflicts of interest

The authors declare no conflicts of interest.

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18 August 2015